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(RESEARCH ARTICLE)

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Effect of Sesame Oil on Cisplatin-Induced Uterine Lesions in Wistar Rats

IKeh Chibuike Anthony [1,*], Abu Sunday ojima [2], Ariomaghwa Loveth Ogheneochuko [3] and Oguamah precious chisom Leonard [4]

[1] *Department of Anatomy, Faculty of Basic Medical Sciences, Bingham University Karu, Nasarawa State, Nigeria.*
[2] *Department of Food Science and Technology, Federal Polytechnic Idah, Kogi state.*
[3] *Department of Nursing, Ladoke Akintola University of Technology Ogbomosho, Oyo State Nigeria.*
[4] *Department of Nursing, university of Port Harcourt, Nigeria.*

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Abstract

Cisplatin is an effective chemotherapeutic agent but can cause reproductive tract toxicity, including uterine injury, potentially mediated by oxidative stress. Sesame oil contains antioxidant bioactive compounds that may mitigate such damage.To evaluate the effect of sesame oil on cisplatin-induced uterine lesions in female Wistar rats using histology, oxidative stress markers, and gonadotropins. Thirty-six female Wistar rats were randomized into four groups (n = 9): control, cisplatin-only, cisplatin + sesame oil (5 mL/kg/day), and cisplatin + sesame oil (6.5 mL/kg/day) for 7 days. Body weight and uterine weight indices were recorded. Uterine MDA and SOD were assessed from tissue homogenates. Serum LH and FSH were measured by ELISA. Uterine sections were stained with H&E and scored semi-quantitatively (0–3) for epithelial disruption, stromal changes, glandular distortion, inflammation, and vascular congestion. Cisplatin increased uterine MDA (0.53 ± 0.04 vs 0.12 ± 0.02) and reduced SOD (3.5 ± 0.3 vs 8.1 ± 0.4) compared with control, and produced higher lesion scores (total 9 vs 0). Sesame oil co-treatment improved oxidative status and histology: 5 mL/kg reduced MDA (0.03 ± 0.01) and improved SOD (7.4 ± 0.5) with a lower lesion score (total 4), while 6.5 mL/kg normalized MDA (0.12 ± 0.02), produced the highest SOD (8.6 ± 0.4), and yielded near-preserved histology (total 1). Sesame oil reduced cisplatin-associated oxidative stress and uterine histological damage, with stronger protection observed at 6.5 mL/kg/day. Hormonal findings, especially markedly elevated FSH in the 5 mL/kg group, require cautious interpretation with unit/dilution verification and consideration of cycle-related variability.

Keywords: Cisplatin; Sesame oil; Uterine toxicity; Oxidative stress; Malondialdehyde; Superoxide dismutase; Follicle-stimulating hormone; Luteinizing hormone

1. Introduction

Cisplatin (cis-diamminedichloroplatinum II) remains a cornerstone chemotherapeutic for a wide range of solid malignancies, including gynecologic cancers, because of its potent cytotoxicity mediated largely through covalent binding to DNA and formation of platinum DNA adducts that disrupt replication and transcription, activate DNA-damage signaling, and ultimately promote apoptosis in rapidly dividing cells [1]. Despite its clinical effectiveness, cisplatin therapy is constrained by significant off-target toxicities (classically nephrotoxicity, neurotoxicity and ototoxicity), with accumulating evidence that oxidative stress, inflammatory signaling, mitochondrial dysfunction and apoptosis contribute substantially to tissue injury and inter-individual variability in toxicity risk [2]. With improving cancer survival rates, preserving female reproductive health has become an increasingly important clinical and translational priority [3,4]. While gonadotoxicity and premature ovarian insufficiency are well recognized adverse outcomes of chemotherapy exposure, reproductive harm is not limited to the ovary; normal fertility and healthy pregnancy depend on coordinated endocrine signaling and intact structure function relationships across the reproductive tract, including

* Corresponding author: IKeh Chibuike Anthony

the uterus [3,5]. Importantly, uterine impairment can compromise implantation and pregnancy outcomes even when ovarian function is partially retained or medically supported, underscoring the need to understand and prevent uterine-specific toxic effects of anticancer regimens [3]. Experimental data indicate that cisplatin exposure can adversely affect both ovarian and uterine tissues, producing biochemical and histopathological changes consistent with reproductive tract injury in rodent models [6,7]. These findings align with broader concerns that anticancer therapies may damage uterine physiology and reduce the capacity to sustain pregnancy, potentially through mechanisms that include altered vascularity, stromal remodeling, and disruption of hormone-responsive endometrial function [3]. However, compared with the extensive literature on cisplatin nephrotoxicity, mechanistic and interventional studies focusing on uterine injury remain relatively limited, leaving an important gap in reproductive toxicology research.

Oxidative stress is increasingly implicated in chemotherapy-related reproductive toxicity and infertility, and redox homeostasis is also central to normal endometrial cycling, decidualization and receptivity [4,8]. In reproductive tissues, excessive reactive oxygen species (ROS) generation and impaired antioxidant defenses can amplify lipid peroxidation, inflammation and apoptosis, thereby disrupting hormone-responsive tissue remodeling [2,4,8]. Malondialdehyde (MDA), commonly quantified by thiobarbituric-acid reactivity assays, is widely used as a proxy marker of lipid peroxidation (with recognized interpretive limitations), while superoxide dismutase (SOD) activity reflects a key enzymatic antioxidant defense against superoxide radicals [9,10]. Together, histopathology integrated with redox biomarkers and endocrine profiling can provide a mechanistically informative assessment of reproductive tract injury and recovery following toxicant exposure.

Sesame (Sesamum indicum L.) seed oil is an edible oil notable for its high content of unsaturated fatty acids and naturally occurring antioxidants, including lignans such as sesamin, sesamolin and their derivatives (e.g., sesamol), which contribute to oxidative stability and biological activity [11,12]. Beyond nutritional relevance, accumulating pharmacological evidence supports antioxidant and anti-inflammatory actions of sesame lignans; sesamin has been extensively discussed for its capacity to modulate oxidative stress and inflammatory pathways, and sesamol has been highlighted as a bioactive lignan with immunomodulatory and anti-inflammatory properties [13,14]. Given that oxidative stress and inflammation are central features of cisplatin toxicity [2,4], sesame oil represents a plausible candidate for mitigating cisplatin-induced reproductive tract injury. Therefore, the present study investigates whether sesame oil can ameliorate cisplatin-induced uterine lesions in Wistar rats, using uterine histology alongside oxidative stress indices and reproductive hormone profiling to evaluate potential protective effects and provide mechanistic insight.

2. Research Methodology

2.1. Study design

This was an experimental study designed to evaluate the protective effect of sesame oil on cisplatin-induced uterine lesions in female Wistar rats. Uterine injury and recovery were assessed using histological evaluation (H&E staining), oxidative stress biomarkers (malondialdehyde [MDA] and superoxide dismutase [SOD]), and serum reproductive hormones (follicle-stimulating hormone [FSH] and luteinizing hormone [LH]). Body weight, absolute uterine weight, and relative uterine weight were also determined.

2.2. Study location

All animal handling, tissue processing, and laboratory analyses were carried out in the Department of Anatomy, Faculty of Basic Medical Sciences, Bingham University, Karu, Nasarawa State, Nigeria.

2.3. Chemicals and reagents

Cisplatin injection (cis-diamminedichloroplatinum II) was purchased from a registered pharmacy in Abuja, Nigeria. Sesame oil was purchased from a retail outlet in Abuja, Nigeria. All other chemicals and reagents used for biochemical estimations were of analytical grade. Cisplatin was administered on a body-weight basis and expressed in mg/kg, determined from the concentration stated on the vial label and the volume administered per kilogram body weight using:

$$Dose(mg/kg) = \text{Volume administered (mL/kg)} \times \text{Concentration (mg/mL)}$$

2.4. Experimental animals and husbandry

Adult female Wistar rats were obtained from an accredited animal facility and acclimatized for at least seven (7) days prior to the commencement of the experiment. Animals were housed in well-ventilated cages under standard laboratory conditions (12-hour light/dark cycle) and fed standard pellet diet with water provided ad libitum. All experimental procedures were conducted in accordance with institutional guidelines for the care and use of laboratory animals, and ethical approval was obtained before the study commenced.

2.5. Experimental grouping and treatment protocol

2.5.1. Animal grouping

Thirty-six (36) adult female Wistar rats were randomly assigned into four experimental groups (n = 9 per group) and treated for seven (7) days as follows:

Group	Treatment	Sesame oil dose	Duration
I	Normal control (normal saline)	–	7 days
II	Cisplatin only (positive control)	–	7 days
III	Cisplatin + sesame oil (low dose)	5 mL/kg/day (oral)	7 days
IV	Cisplatin + sesame oil (high dose)	6.5 mL/kg/day (oral)	7 days

Sesame oil was administered orally using a gavage cannula. Cisplatin was administered under aseptic conditions using the route adopted for this experimental model.

2.5.2. Randomization and bias control

Animals were allocated to groups using simple randomization. Histological assessment, lesion scoring, and selection of representative photomicrographs were carried out with measures to reduce observer bias.

2.5.3. Body weight and uterine weight measurements

Body weights were recorded at baseline (Day 0) and at the end of treatment (Day 7) using a calibrated weighing balance.

2.5.4. Uterine weight and relative uterine weight

After sacrifice, the uterus was excised, carefully trimmed of surrounding fat and connective tissue, blotted dry, and weighed to obtain absolute uterine weight (g). Relative uterine weight was calculated as:

Relative uterine weight (%) = (uterine weight (g) / final body weight (g)) × 100

2.5.5. Sample collection and preparation

Following the final administration, animals were anesthetized and blood was collected into plain tubes. Blood samples were allowed to clot and centrifuged at 3000 rpm for 10–15 minutes to obtain serum for hormonal assays (LH and FSH). Uterine tissues were harvested and divided into two portions: one portion was processed for biochemical assays, and the second portion was fixed for histology.

2.5.6. Preparation of uterine homogenate

For oxidative stress assays, uterine tissue samples were rinsed in ice-cold normal saline, weighed, and homogenized in cold phosphate buffer (pH 7.4) to obtain a 10% (w/v) homogenate. The homogenate was centrifuged at 3000 rpm for 15–30 minutes at 4°C, and the supernatant was collected for biochemical estimations.

2.5.7. Determination of malondialdehyde (MDA)

Uterine MDA levels were estimated using the thiobarbituric acid reactive substances (TBARS) method. Briefly, tissue supernatant was reacted with thiobarbituric acid under acidic conditions and heated to form a pink chromogen. Absorbance was measured at 532 nm using a spectrophotometer. MDA concentration was calculated using an appropriate extinction coefficient and expressed on a consistent basis.

2.5.8. Determination of superoxide dismutase (SOD)

SOD activity in uterine homogenate was determined using a standard spectrophotometric method based on the ability of SOD to inhibit superoxide-mediated reactions. Results were expressed consistently as enzymatic activity per tissue basis.

2.5.9. Determination of serum LH and FSH

Serum LH and FSH were quantified using enzyme-linked immunosorbent assay (ELISA) kits following the manufacturer's instructions. Absorbance was measured using a microplate reader at the recommended wavelength, and hormone concentrations were derived from standard calibration curves. Results were reported in the units specified by the assay kits.

2.5.10. Histological processing and photomicrography

Uterine tissue samples designated for histology were fixed in 10% neutral buffered formalin for 24–48 hours, processed routinely, embedded in paraffin wax, sectioned at 4–5 µm, and stained with hematoxylin and eosin (H&E). Stained sections were examined under a light microscope and representative photomicrographs were captured at ×100 and ×400 magnifications.

2.6. Histological lesion scoring and image annotation

2.6.1. Lesion scoring

To objectively demonstrate uterine distortions and tissue injury, uterine sections were scored using a semi-quantitative grading system (0–3) for each of the following features:

- Endometrial epithelial disruption/degeneration (0 = none to 3 = severe)
- Stromal hyperplasia and/or edema (0 = none to 3 = severe)
- Glandular distortion/dilation (0 = none to 3 = severe)
- Inflammatory cell infiltration (0 = none to 3 = severe)
- Vascular congestion and/or hemorrhage (0 = none to 3 = severe)

A total lesion score was computed for each animal by summing domain scores, and mean lesion scores were compared across groups.

2.6.2. Image labeling (minimum standard)

Representative photomicrographs were annotated to show key uterine structures: lumen (L), endometrium (En), endometrial glands (G), stroma (S), and myometrium (My). Arrows/asterisks were used to highlight pathological features such as epithelial degeneration, stromal disruption, edema, inflammatory infiltration, glandular distortion, and vascular congestion. Magnification was indicated on each image, and a scale bar was included where possible.

2.7. Statistical analysis

Data were analyzed using standard statistical procedures. Results were expressed as mean ± SEM. Differences among groups were assessed using one-way ANOVA followed by an appropriate post-hoc multiple comparison test. Statistical significance was set at $p < 0.05$.

3. Results

3.1. Summary of groups and measured outcomes

Thirty-six (36) adult female Wistar rats were randomly assigned into four groups (n = 9/group): Group I (Normal Control), Group II (Cisplatin-only), Group III (Cisplatin + sesame oil 5 mL/kg), and Group IV (Cisplatin + sesame oil 6.5 mL/kg). Outcome measures included body weight changes, uterine weight indices, uterine oxidative stress biomarkers (MDA and SOD), serum gonadotropins (LH and FSH), and uterine histopathological lesion scores.

3.1.1. Body weight parameters

Baseline body weights (Day 0) were comparable across groups, ranging from 187.9 ± 4.8 g to 189.5 ± 5.1 g (Table 1), indicating that the animals were relatively well matched before treatment. After seven days, the control group (Group

I) gained weight from 188.2 ± 4.3 g to 198.4 ± 4.7 g, representing a mean increase of +10.2 g (+5.4%). In contrast, the cisplatin-only group (Group II) showed a slight reduction in weight from 189.5 ± 5.1 g to 186.3 ± 5.0 g, corresponding to a mean change of −3.2 g (−1.7%), suggesting a mild systemic effect of cisplatin exposure.

Sesame oil co-treatment improved weight trajectory relative to the cisplatin-only group. Group III (cisplatin + sesame oil 5 mL/kg) increased from 187.9 ± 4.8 g to 192.1 ± 5.1 g (+4.2 g; +2.2%), while Group IV (cisplatin + sesame oil 6.5 mL/kg) increased from 188.7 ± 4.6 g to 196.8 ± 5.2 g (+8.1 g; +4.3%). Overall, the high-dose sesame oil group demonstrated weight recovery closer to the control group than the low-dose sesame oil group (Table 1).

Table 1 Body weight parameters (Mean ± SEM)

Group	n	Day 0 (g) Mean ± SEM	Day 7 (g) Mean ± SEM	Weight change (g)	% change
I (Control)	9	188.2 ± 4.3	198.4 ± 4.7	+10.2	+5.4%
II (Cisplatin)	9	189.5 ± 5.1	186.3 ± 5.0	−3.2	−1.7%
III (Cis + SO 5 mL/kg)	9	187.9 ± 4.8	192.1 ± 5.1	+4.2	+2.2%
IV (Cis + SO 6.5 mL/kg)	9	188.7 ± 4.6	196.8 ± 5.2	+8.1	+4.3%

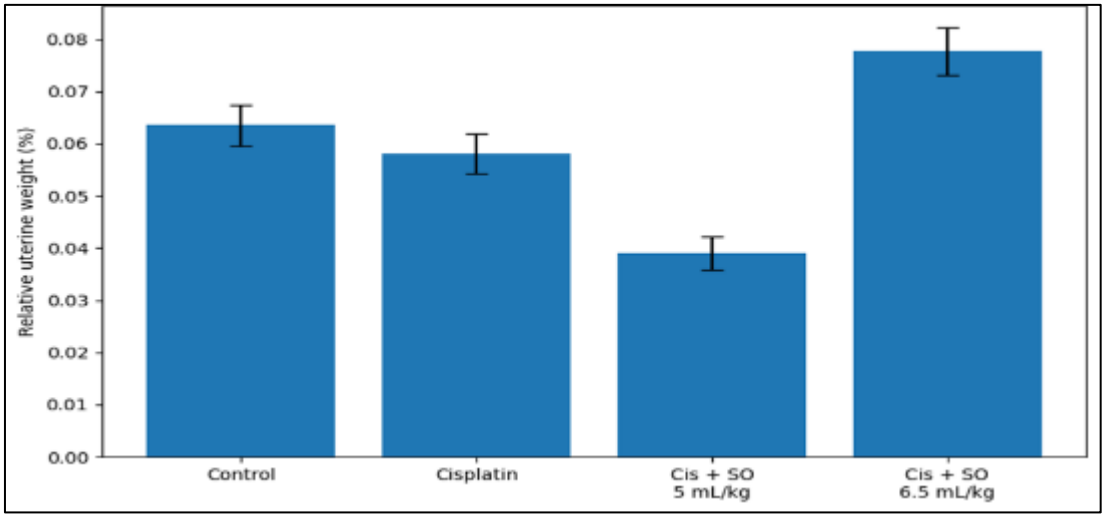

Figure 1 Relative uterine weight (%) across experimental groups

Shows uterus-to-body-weight proportion; lowest in Cis + SO 5 mL/kg and highest in Cis + SO 6.5

3.1.2. Uterine weight indices

Absolute uterine weight was highest in the control group (0.126 ± 0.008 g). The cisplatin-only group showed a reduction in uterine weight to 0.108 ± 0.007 g, suggesting cisplatin-associated reduction in uterine mass (Table 2). Among sesame oil-treated groups, Group III recorded the lowest uterine weight (0.075 ± 0.006 g), whereas Group IV recorded the highest (0.153 ± 0.009 g), exceeding the control mean. This pattern was mirrored in relative uterine weight.

Relative uterine weight (%) in the control group was 0.0635 ± 0.0040%. The cisplatin-only group showed a slight reduction to 0.0580 ± 0.0038%. Group III exhibited the lowest relative uterine weight (0.0390 ± 0.0031%), while Group IV showed the highest relative uterine weight (0.0777 ± 0.0046%) (Table 2). These results indicate that sesame oil at 6.5 mL/kg was associated with an increased uterus-to-body-weight proportion compared to cisplatin-only and control groups.

Table 2 Uterine weight indices (Mean ± SEM)

Group	n	Absolute uterus weight (g)	Relative uterus weight (%)
I (Control)	9	0.126 ± 0.008	0.0635 ± 0.0040
II (Cisplatin)	9	0.108 ± 0.007	0.0580 ± 0.0038
III (Cis + SO 5 mL/kg)	9	0.075 ± 0.006	0.0390 ± 0.0031
IV (Cis + SO 6.5 mL/kg)	9	0.153 ± 0.009	0.0777 ± 0.0046

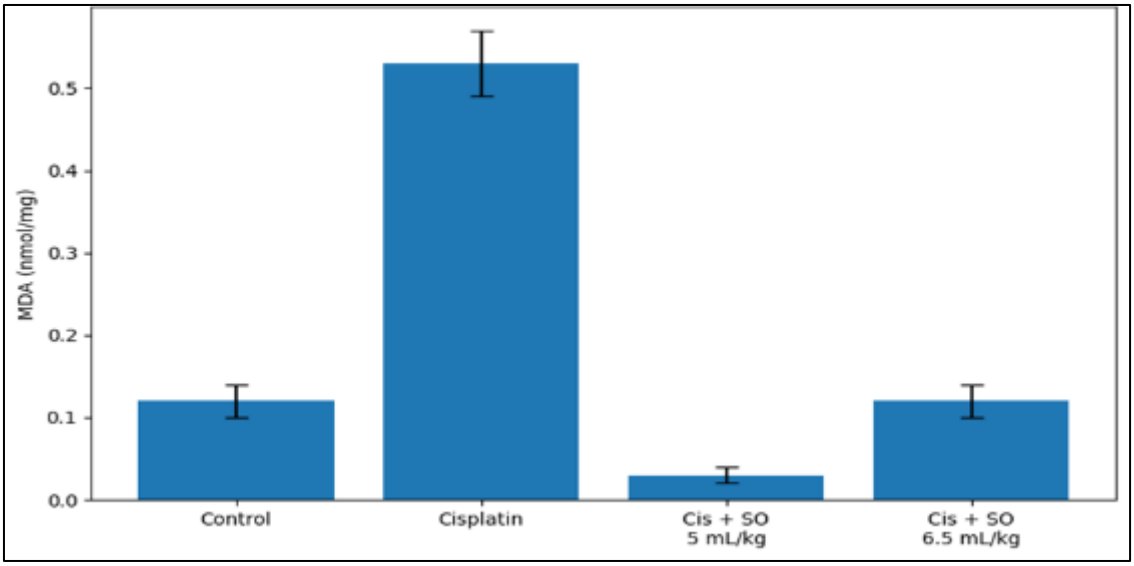

Figure 2 Uterine malondialdehyde (MDA) across experimental groups

Shows lipid peroxidation; cisplatin markedly increased MDA, while sesame oil reduced it (strongest reduction with co-treatment).

3.1.3. Uterine oxidative stress parameters (MDA and SOD)

Cisplatin administration produced a clear oxidative stress profile in uterine tissue (Table 3). Uterine MDA level in the cisplatin-only group increased markedly to 0.53 ± 0.04 nmol/mg, compared with the control group value of 0.12 ± 0.02 nmol/mg, indicating elevated lipid peroxidation. Conversely, uterine SOD activity was reduced in the cisplatin-only group to 3.5 ± 0.3 U/mg, compared with 8.1 ± 0.4 U/mg in controls, demonstrating reduced antioxidant defense in the cisplatin-treated animals. Sesame oil co-treatment improved redox balance. Group III showed a marked reduction in MDA to 0.03 ± 0.01 nmol/mg, alongside improved SOD activity (7.4 ± 0.5 U/mg). Group IV demonstrated MDA level comparable to control (0.12 ± 0.02 nmol/mg) and the highest SOD activity among all groups (8.6 ± 0.4 U/mg) (Table 3). These results suggest that sesame oil substantially attenuated cisplatin-induced oxidative damage in the uterus, with a stronger normalization of antioxidant status observed at the 6.5 mL/kg dose.

Table 3 Oxidative stress parameters (Mean ± SEM)

Group	n	MDA (nmol/mg) Mean ± SEM	SOD (U/mg) Mean ± SEM
I (Control)	9	0.12 ± 0.02	8.1 ± 0.4
II (Cisplatin)	9	0.53 ± 0.04	3.5 ± 0.3
III (Cis + SO 5 mL/kg)	9	0.03 ± 0.01	7.4 ± 0.5
IV (Cis + SO 6.5 mL/kg)	9	0.12 ± 0.02	8.6 ± 0.4

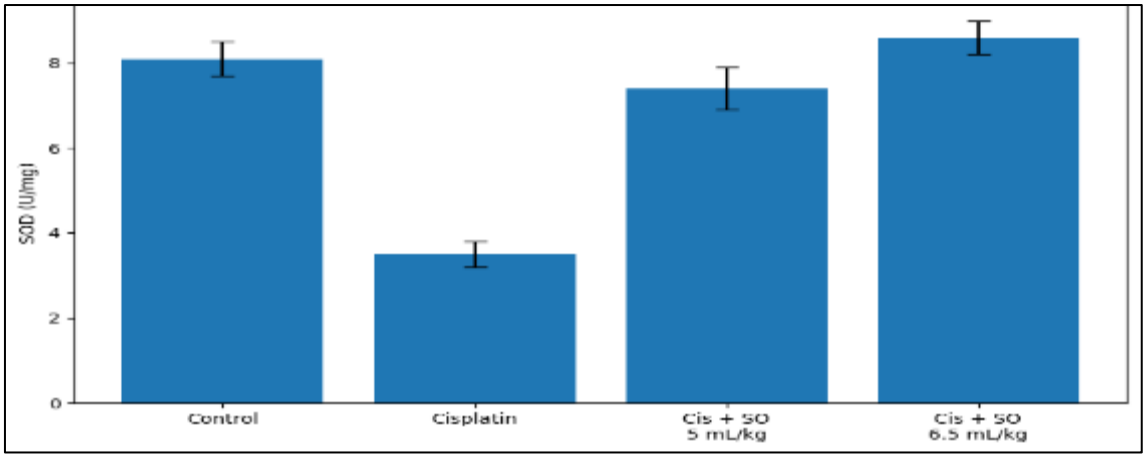

Figure 3 Uterine superoxide dismutase (SOD) activity across experimental groups

Shows antioxidant defense; cisplatin reduced SOD, while sesame oil restored/increased SOD, highest at 6.5 mL/kg.

3.1.4. Serum gonadotropins (LH and FSH)

Serum LH concentration in the control group was 1.2 ± 0.1 IU/L, while the cisplatin-only group showed a similar value (1.1 ± 0.1 IU/L), indicating minimal cisplatin effect on LH at the measured time point (Table 4). In the sesame oil co-treated groups, LH increased markedly in Group III to 10.2 ± 0.6 IU/L, while Group IV showed a smaller elevation to 2.5 ± 0.2 IU/L (Table 4). Thus, the low-dose sesame oil group exhibited the highest LH concentration. FSH levels in the control group were 1.0 ± 0.1 IU/L, increasing to 6.0 ± 0.4 IU/L in the cisplatin-only group, suggesting that cisplatin exposure was associated with increased FSH secretion relative to control. Co-treatment with sesame oil was associated with substantially higher FSH levels: Group III recorded 100.0 ± 4.8 IU/L, while Group IV recorded 35.0 ± 2.5 IU/L (Table 4). Overall, sesame oil co-treatment was associated with marked alterations in gonadotropin levels, with Group III showing the highest elevations.

Table 4 Serum gonadotropins (Mean ± SEM)

Group	n	LH (IU/L) Mean ± SEM	FSH (IU/L) Mean ± SEM
I (Control)	9	1.2 ± 0.1	1.0 ± 0.1
II (Cisplatin)	9	1.1 ± 0.1	6.0 ± 0.4
III (Cis + SO 5 mL/kg)	9	10.2 ± 0.6	100.0 ± 4.8
IV (Cis + SO 6.5 mL/kg)	9	2.5 ± 0.2	35.0 ± 2.5

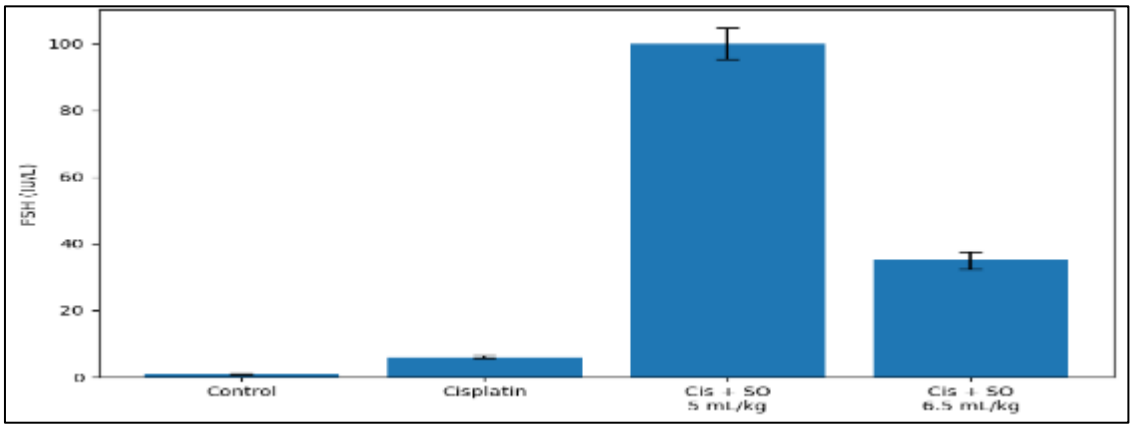

Figure 4 Serum follicle-stimulating hormone (FSH) across experimental groups

Shows gonadotropin response; FSH increased with cisplatin and was markedly higher in sesame oil co-treated groups, especially 5 mL/kg.

3.1.5. Uterine histopathology and lesion scoring

Histopathological assessment of uterine sections from the control group (Group I) showed normal uterine histoarchitecture with intact endometrial epithelium, well-formed endometrial glands, orderly stromal organization, and normal myometrial structure (Figure 6). In contrast, uterine sections from cisplatin-treated rats (Group II) demonstrated uterine injury, including epithelial disruption/degeneration, stromal disorganization with evidence of edema and inflammatory cell infiltration, glandular distortion, and vascular congestion (Figure 7). Consistent with these findings, lesion scoring in Group II showed moderate injury with scores of 2 for epithelium, glands, stroma, and congestion, and 1 for inflammation, producing a total lesion score of 9 (Table 5). Sesame oil co-treatment reduced lesion severity. Group III exhibited milder changes compared with Group II, with lesion scores of 1 for epithelium, glands, and stroma, 0 for inflammation, and 1 for congestion, resulting in a total lesion score of 4 (Table 5). Histologically, Group III showed partial preservation of uterine architecture with reduced epithelial disruption and less stromal damage compared to cisplatin-only animals (Figure 8).

Group IV demonstrated near-preservation of uterine architecture. Lesion scoring showed 0 for epithelium, glands, inflammation, and congestion, and 1 for stroma, giving the lowest total lesion score among treated animals (1) (Table 5). Histologically, Group IV sections showed relatively preserved endometrial lining, maintained glandular structure, and largely normal myometrium, with only mild stromal alterations (Figure 9).

Table 5 Uterine histological lesion scoring (0–3 scale)

Group	n	Epithelium	Glands	Stroma	Inflammation	Congestion	Total
I (Control)	9	0	0	0	0	0	0
II (Cisplatin)	9	2	2	2	1	2	9
III (Cis + SO 5 mL/kg)	9	1	1	1	0	1	4
IV (Cis + SO 6.5 mL/kg)	9	0	0	1	0	0	1

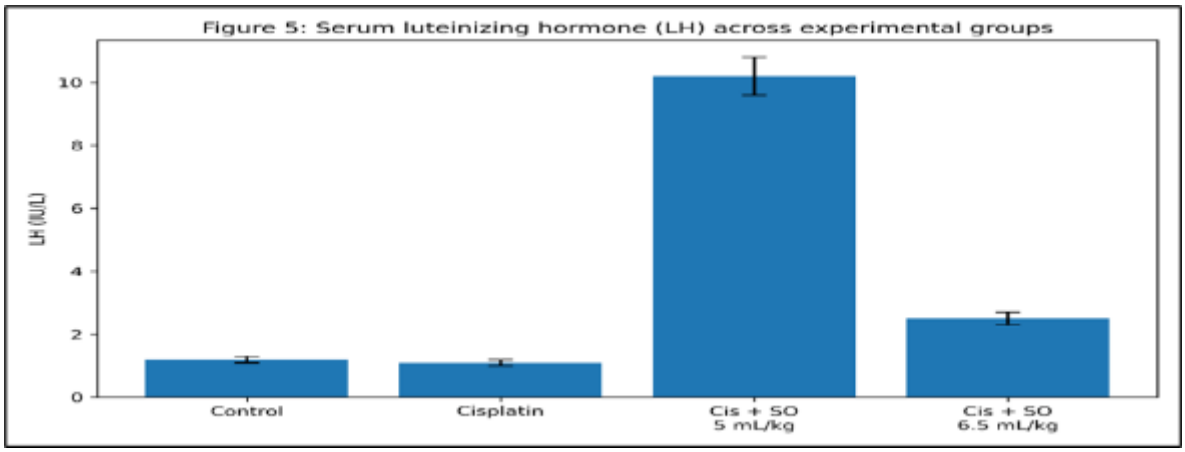

Figure 5 Serum luteinizing hormone (LH) across experimental groups

Shows LH changes; LH was similar in control vs cisplatin-only but increased in sesame oil co-treated groups, highest at 5 mL/kg.

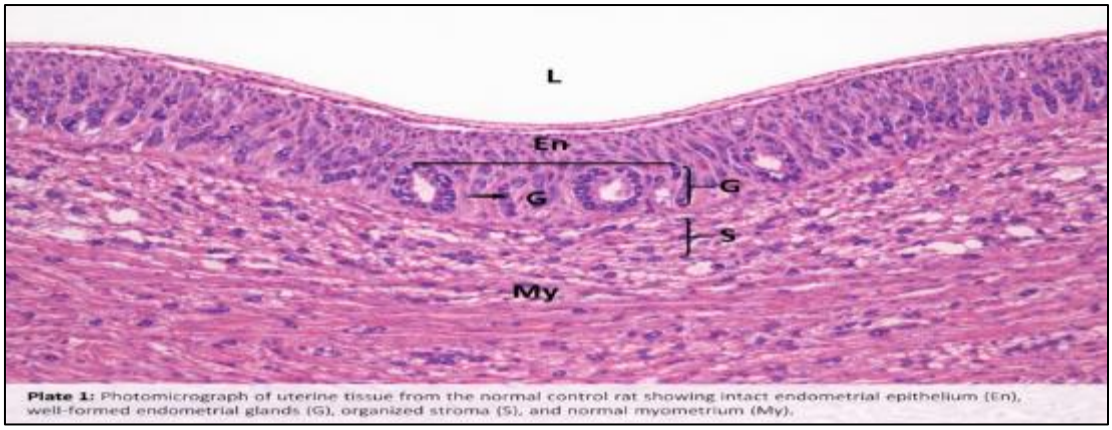

Figure 6 Photomicrograph of uterine tissue from the normal control rat showing intact endometrial epithelium, well-formed endometrial glands, organized stroma, and normal myometrium (H&E, ×100)

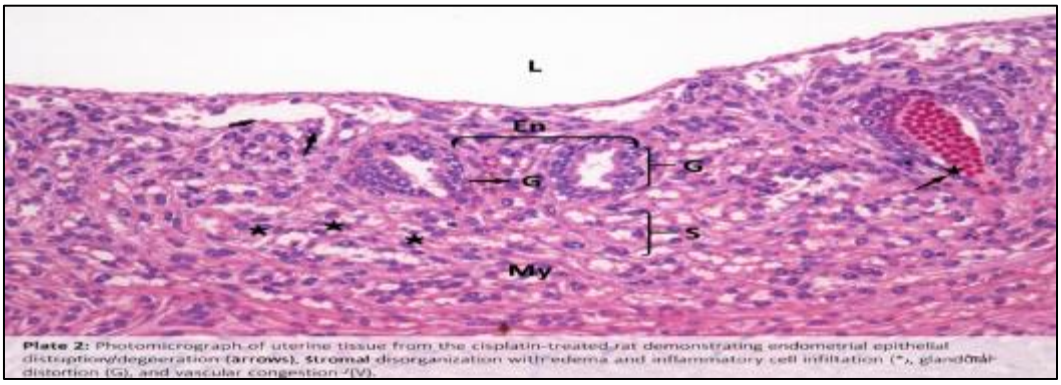

Figure 7 Photomicrograph of uterine tissue from the cisplatin-treated rat demonstrating endometrial epithelial disruption/degeneration, stromal disorganization with edema/inflammatory cell infiltration, glandular distortion, and vascular congestion (H&E, ×100)

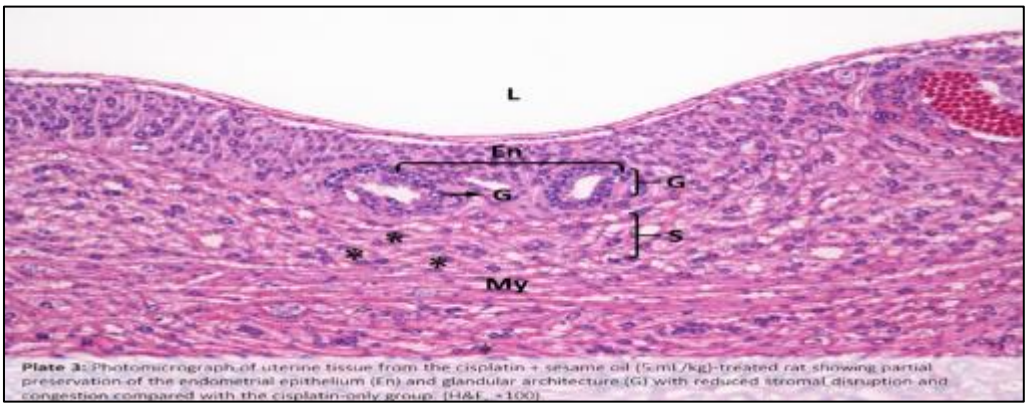

Figure 8 Photomicrograph of uterine tissue from the cisplatin + sesame oil (5 mL/kg) treated rat showing partial preservation of the endometrial epithelium and glandular architecture with reduced stromal disruption compared with the cisplatin-only group (H&E, ×100)

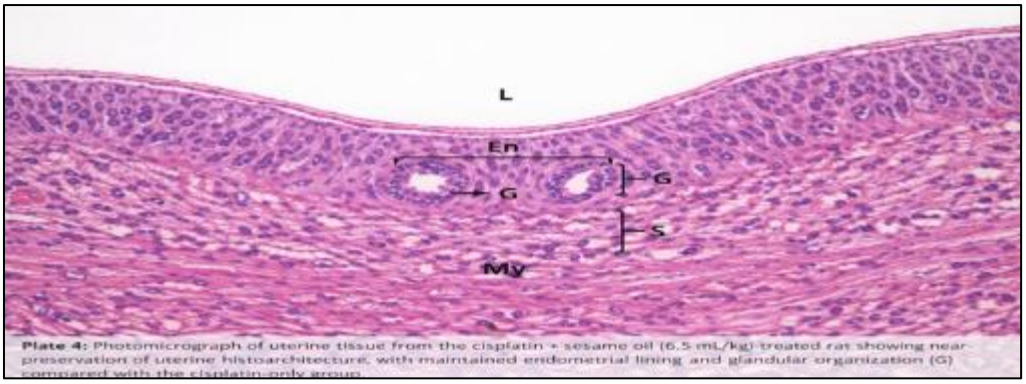

Figure 9 Photomicrograph of uterine tissue from the cisplatin + sesame oil (6.5 mL/kg) treated rat showing near-preservation of uterine histoarchitecture with maintained endometrial lining and glandular organization compared with the cisplatin-only group (H&E, ×100)

4. Discussion

The present study evaluated the effect of sesame oil on cisplatin-induced uterine injury in female Wistar rats using histopathology, oxidative stress biomarkers (MDA and SOD), and gonadotropins (LH and FSH). The major findings were that cisplatin exposure was associated with (i) mild systemic impact reflected by reduced weight gain, (ii) increased uterine oxidative stress (higher MDA and lower SOD), and (iii) histological distortions characterized by epithelial disruption, stromal disorganization, glandular distortion, and vascular congestion. Co-treatment with sesame oil improved redox status and reduced histological lesion severity, with the higher dose (6.5 mL/kg) producing the most consistent protective pattern.

4.1. Cisplatin-induced uterine injury and oxidative stress

Cisplatin is widely reported to induce tissue toxicity through oxidative stress, mitochondrial dysfunction, inflammatory signaling, and apoptosis. In this study, uterine MDA rose markedly in cisplatin-treated rats (0.53 ± 0.04) compared with controls (0.12 ± 0.02), while SOD activity declined (3.5 ± 0.3 vs 8.1 ± 0.4). This pattern supports the interpretation that cisplatin promoted lipid peroxidation and weakened endogenous antioxidant defense in uterine tissue. Histological findings in the cisplatin-only group (epithelial degeneration/disruption, stromal disruption/edema, inflammatory infiltration, glandular distortion, and congestion) align with oxidative–inflammatory tissue injury that can disrupt endometrial integrity and uterine receptivity.

4.2. Protective effects of sesame oil on uterine redox balance and morphology

Sesame oil contains antioxidant constituents (including lignans and other bioactive compounds) that can reduce lipid peroxidation and support antioxidant enzyme activity. In the present study, sesame oil co-treatment substantially improved uterine redox indices. The 5 mL/kg group showed a marked reduction in MDA (0.03 ± 0.01) with improved SOD (7.4 ± 0.5), while the 6.5 mL/kg group normalized MDA to control levels (0.12 ± 0.02) and produced the highest SOD activity (8.6 ± 0.4). These biochemical improvements were mirrored by histopathology: lesion scores reduced from 9 (cisplatin-only) to 4 (5 mL/kg) and 1 (6.5 mL/kg), indicating that sesame oil mitigated structural distortion and preserved uterine architecture, particularly at the higher dose.

4.3. Body weight and uterine weight patterns

Cisplatin-only animals exhibited slight weight loss, while controls gained weight, suggesting a systemic effect of cisplatin during the study period. Sesame oil improved weight recovery, most notably at 6.5 mL/kg. Uterine weight patterns were more complex: cisplatin reduced uterine weight relative to control, consistent with tissue injury and reduced uterine mass. The high-dose sesame oil group showed increased uterine weight and higher relative uterine weight, which when interpreted alongside improved histology likely reflects preservation/recovery of tissue integrity. However, uterine weight can also be influenced by physiological cycling and stromal water content; therefore, pairing weight indices with histology (as done here) is essential.

4.4. Gonadotropin alterations and interpretation of the very high FSH value

Gonadotropin measurements showed major between-group differences, particularly the high LH and FSH values in the 5 mL/kg group. Because gonadotropins fluctuate with estrous cycling and are strongly influenced by ovarian feedback, these changes may reflect disruption and/or compensation within the hypothalamic pituitary gonadal axis in response to cisplatin injury and antioxidant intervention. Importantly, the markedly elevated FSH reported in Group III (100.0 ± 4.8) is unusually high and should be interpreted cautiously. Such a value warrants confirmation of ELISA kit units, calibration range, and any dilution factor applied during analysis. Where unit reporting is correct, the finding may indicate pronounced endocrine perturbation; where unit conversion or dilution correction errors exist, the values should be recalculated and reported strictly in kit-specified units.

4.5. Limitations and future directions

The study duration (7 days) may not fully represent repeated chemotherapy cycles. Hormonal interpretation is limited without explicit estrous-cycle staging, and the absence of a sesame-oil-only group limits conclusions about sesame oil's independent endocrine effects. In addition, TBARS-based MDA measurement is a commonly used index of lipid peroxidation but has known specificity limitations. Future work should incorporate estrous staging, a sesame-oil-only control, and molecular markers of inflammation and apoptosis (e.g., cytokines and caspase pathways), as well as ovarian reserve indices, to strengthen mechanistic inference and reproductive relevance.

The data support that cisplatin induces oxidative stress–linked uterine injury and that sesame oil co-treatment attenuates oxidative damage and reduces histological lesions, with the higher dose showing the most consistent tissue protection.

5. Conclusion

Cisplatin exposure induced uterine injury in female Wistar rats, evidenced by elevated lipid peroxidation (increased MDA), reduced antioxidant defense (decreased SOD), and histological distortions including epithelial degeneration, stromal disruption, glandular distortion, and vascular congestion. Sesame oil co-treatment improved uterine antioxidant status and reduced lesion severity, with 6.5 mL/kg showing the most marked preservation of uterine histoarchitecture. These findings suggest sesame oil has potential protective effects against cisplatin-induced uterine toxicity, likely mediated in part through antioxidant mechanisms. Further studies incorporating estrous-cycle control, additional endocrine validation, and molecular injury markers are recommended.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare that they have no conflict of interest.

Statement of ethical approval

At the time this study was conducted, Bingham University, Karu, Nasarawa State, Nigeria did not operate a formal animal ethics review committee and no formal ethical clearance number was issued. However, the study protocol was reviewed and permitted by the Head of Department, Department of Anatomy, Faculty of Basic Medical Sciences, Bingham University, Karu, Nasarawa State, Nigeria, on August, 2020. All animal procedures were carried out in accordance with institutional standards for the care and use of laboratory animals, with efforts made to minimize pain, distress, and discomfort to the animals.

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